(VASCULAR MEDICINE (NON-CORONARY): CLINICAL SCIENCE)

41. The myocardial protective effect of dexmedetomidine in high risk patients undergoing aortic vascular surgery

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Dexmedetomidine provides perioperative cardiac protection in high risk patients assessment the effect of dexmedetomidine in high risk patients undergoing aortic vascular surgery. Arandomized study included 150 patients classified into two groups($n = 75$). Group D: The patients received a loading dose of 1 μg/kg dexmedetomidine over 15 min before induction and maintained as an infusion of 0.3 μg/kg/hr to the end of the procedure. Group C: The patients received an equal volume of normalsaline. The dexmedetomidine decreased heart rate and minimized the changes in blood pressure compared to control group ($p < 0.05$). Also, it decreased the incidence of myocardial is chemia reflected by troponin I level and ECG changes($p < 0.05$). Dexmedetomidine decreased the requirement for nitroglycerine and norepinephrine compared to control group ($p < 0.05$). The incidence of hypotension and bradycardia were significantly higher with dexmedetomidine ($p < 0.05$). The dexmedetomidine is safe and effective in patients undergoing aortic vascular surgery. It decreases the changes in heart rate and blood pressure during the procedures. It provide scardiac protection in high risk patients reflected by decreasing the incidence of myocardial ischemia and serum level of troponin. The main side effects of dexmedetomidine were hypotension and bradycardia.

http://dx.doi:10.1016/j.jsha.2016.04.042

Microcirculation and Cerebral/Coronary/peripheral circulation

42. The effect of ticagrelor on coronary blood after primary PCI when compared with clopidogril

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Primary PCI (PPCI) has been established as the best treatment for acute MI when it is used appropriately. It is known to give better TIMI III flow and better frame count when compared with thrombolytics. Loading with P2Y12 inhibitors in the ER prior to primary PCI is an important step in antiplatelet therapy for acute myocardial infarction. In this study We report the effect of loading with two different P2Y12 inhibitors (ticagrelor and clopidogril) on the TIMI frame count in the culprit artery after successful PPCI. Ticagrelor may affect coronary microcirculation and coronary blood flow through faster and stronger platelet inhibition. We randomized 44 patient who presented to our center with acute MI into two groups. The first group received a loading with 180mg of ticagrelor and the second group received a loading with 600 mg of clopidogril. The mean door to balloon time was 98 ± 12 min. All patients in both groups received a loading with 162 mg of aspirin. GP IIb/IIIa inhibitors were used in all cases together with adjusted dose heparin. Stent usage was 100%. No thrombectomy or thrombus aspiration device was used in any of these cases. TIMI III flow after stenting was achieved in all culprit arteries. Then we calculated the TIMI frame count in the culprit artery after successful primary PCI. The mean corrected TIMI frame count in the culprit artery post PCI was 18.34 ± 3.16 frames in group 1 (Ticagrelor group) and 28.73 ± 3.92 in group 2 (clopidogril group) ($p = 0.02$). Loading with ticagrelor gives faster flow after successful primary PCI in the culprit artery of acute MI when compared with clopidogril. This can be explained by the fact that ticagrelor therapy give faster P2Y12 inhibition thus faster antiplatelet therapy causing less platelet aggregation resulting in less distal embolization and reduced production of inflammatory mediators and adhesion molecules which may result in faster restoration of normal endothelial function. This finding may partially explain the mortality benefit of ticagrelor in a previous ACS study. A larger prospective randomized study is needed to confirm this finding.

http://dx.doi:10.1016/j.jsha.2016.04.043

Cellular biology function

LIGAND-MEDIATED SIGNALING AND RECEPTOR PHARMACOLOGY

43. Calmodulin regulating calcium sensitivity of Na channels

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By extrapolating information from existing research and observing previous assumptions regarding the structure of the Na Channel, this experiment was conducted under the hypothesis that the Na Channel is in part regulated by the calmodulin protein, as a result proving calcium sensitivity of the Na Channel. Furthermore, we assume that there is a one to one stoichiometry between the Na Channel and the Calmodulin. There has been extensive research into the functionality and structure of sodium ion channels (Na channels), as several diseases are associated with the lack of regulation of sodium ions, that is caused by the disfunction of these Na channels. However, one highly controversial matter in the field is the importance of the protein calmodulin (CaM) and calcium in Na channel function. Calmodulin is a protein that is well known for its role as a calcium binding messenger protein, and that association is believed to play an indirect role in regulating the Na chan-

nel through the Na channel's supposed calcium sensitivity. While there are proponents for both sides, there has been relatively little research that provides strong evidence for either case. In this experiment, the effect of calmodulin on NaV 1.5 is tested by preparing a set of cardiac cells (of the human specie) with the NaV 1.5 C-Termini and CaM protein, which were then to be placed in solutions with varying concentrations of calcium. We took special care to test multiple concentrations of calcium, as previous studies have tested very low concentrations, with Manu Ben-Johny's team from the John Hopkins laboratory in particular testing up to a meager 50 micromolar, despite producing a well-respected paper (By comparison, the average Na channel can naturally sustain a concentration of almost 1-2 millimolar and on some occasions, reaching even higher concentrations). After using light scattering and observing the signals given off by the calcium interacting with these Nav1.5/CaM complexes across the varying calcium concentrations, the overall pattern indicated that there was a one to one stoichiometry between calmodulin and Nav 1.5. More importantly, it indicated calcium sensitivity of the Na channel. With this research, a definitive answer has been drawn regarding the importance of calmodulin in calcium modulation in Na channels. Not only does this have the effect of creating a foundation for further research into the structure and function of Na channels, but it also gives deep insight into fundamental functions of the channel that can play a major role into the creation of drugs to treat the many cardiac diseases associated with dysfunction of the channel.

http://dx.doi:10.1016/j.jsha.2016.04.044

Chronic and acute ischemic heart disease

BIOMARKERS

44. Copeptin as early marker of acute non-ST elevation myocardial infarction in patients suspected with acute coronary syndrome

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Rapid diagnosis and management of AMI have great impact on morbidity and mortality. Diagnosis which is based on elevation of cardiac biomarkers has its limitations. Copeptin is the C-terminal part of the vasopressin prohormone. The pathophysiology mode of release should theoretically add diagnostic information of cardiac cell necrosis. One of the major limitations of cardiac biomarkers is the delayed release in circulation. So looking for a new marker with a short diagnostic time window is needed. Aim is to determine the role of copeptin as an early marker for acute non-ST elevation MI (NSTEMI). This study included 88 patients with chest pain. They were divided into 2 groups. Group (1); included 30 patients with diagnosis of NSTEMI. Diagnosis of AMI was established according to the universal definition of MI. Group (2); included 58 patients with diagnosis of unstable angina (UA). Full medical history, physical examination, 12 lead ECG, random blood glucose level, renal function, total cholesterol, triglyceride, cardiac troponin I and Copeptin were obtained on admission. Follow up cardiac troponin I was done. Inclusion criteria: Defined as chest pain of $\leqslant$6 h duration since onset, suggestive of myocardial ischemia, and lasting >20 min. at rest. Exclusion criteria: Patients with positive First cardiac troponin were rolled out, patients with ST segment elevation were rolled out. Other exclusion criteria: Patients presenting after a cardiac arrest, Trauma or major surgery within the last 4 week; pregnancy; IV drug abuse; age less than 18 years; shock and sepsis. Patients who were included had second troponin I re- done and copeptin analysis done. In group 1 (NSTEMI) 28 patients had ECG changes and only 2 had NSTEMI without ECG changes. In group 2 (UA) 23 patients had ECG changes and 35 patients had normal ECG. Males and females were 49 and 39. Age in G1 and G2 was 60 ± 4 and 53 ± 5. Copeptin analysis was done 6 h after Infarction or chest pain. All the patients with NSTEMI (30) had positive copeptin and positive troponin except one only who had + troponin only and another one who had + copeptin only. Of the 58 patients without MI none had the two tests positive, only one had + troponin and one had + copeptin. Using ROC curve: copeptin had sensitivity 100% and specificity 82.8% with using cut off point 13.2 pmol/l. So copeptin can be used for early detection of myocardial infarction. Copeptin seems to be an ideal confirmatory marker for rapid rule out of AMI. If the two tests (with troponin) are positive, this is evident MI; if the two are negative it rules out MI.

http://dx.doi:10.1016/j.jsha.2016.04.045

Chronic ischemic heart disease

45. Ezetimibe and statins yields on silent holter ambulatory myocardial ischemia

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Further cholestrol lowering may affect silent ischemia detected on holter monitoring. Cholesterol lowering is associated with a reduction in cardiovascular morbidity and mortality. Statins are the main drugs for cholesterol lowering. Ezetimibe when added to statins gives further reduction in cholesterol but its long-term effect on cardiovascular morbidity and mortality and ischemic events is not known. This study sought to determine whether further cholesterol lowering with ezitimibe will also results in a reduction of myocardial ischemia during daily